1907 Dehon, M., and Brasseur, A.: One case, Écho jéd. du nord., Lille, 1907, xi, 80.

1907 Armone, G.: Two cases. Med. ital., Napoli, 1907, v, 381.

1908 Talley, D. D., Jr.: One case. Old Dominion Jour. Med. and S. Richmond, 1908, vii, 96, 1 pl.

1908 West, S.: One case. Lancet, London, 1908, i, 489.

1908 Holmes, E. B.: One case. New York Med. Jour., 1908, lxxxvii, 786.

1908 Schwarz, E.: One case. Wien. med. Wchnschr., 1908, lviii, 1177; 1248; 1307.

1909 Lasnier, A.: One case. Bull. et mém. soc. anat. de Paris, 1909, lxxxiv, 129.

1909 Josué, O., and Paillard, H.: One case. Bull. et mém. soc. de hôp. de Paris, 1909, 3 s xxviii, 97.

1909 Derscheid: One case. Policlin., Brux., 1909, xviii, 161.

1909 Bergman, W.: One case. Wien. u. Leipz., 1908, 342.

1910 von Reuss, A.: One case. Mitt. d. Gesellsch. f. inn. Med. u. Kinderh. in Wien, 1910, ix, 221.

1911 Manec, M. L.: One case. Bordeaux, 1911, A. Saugnac, 69, p. 80.

1911 Griebel, Hermann: One case. Kiel, 1911, H. Fiencke, 21, p. 80.

1915 Israel, Rosenthal: One case. Ugeskr. f. Laeger, Kobenk, 1915, lxxvii, 1412.

1915 Laurie, H., and Colville, H. C.: One case. Med. Jour., Australia, Sydney, 1915, i, 145.

1917 Hadley, E. C.: One case. Clin. Jour., London, 1917, lxvi, 267.

1919 Christian, H. A.: One case. Med. Clin. North America, Philadelphia, 1919, ii, 1255.

---

# CURVES OF SUGAR AND UREA AFTER STANDARD PROTEIN MEALS.

By Aage Th. B. Jacobsen, M.D.,

and

Harold Edwards,

NEW YORK.

(From the Clinic of Dr. Frederick M. Allen, New York City.)

One of the writers[1] previously reported observations of the effects of diet on the blood sugar of normal persons and diabetics. The results showed no demonstrable influence of protein in the type of cases studied. Rolly and Oppermann[2] observed a rise of blood sugar after protein meals in some diabetics. Strouse, Stein and Wisely[3] found no evidence of a rise of blood sugar in normal persons after carbohydrate-free meals. In animals with suitably severe diabetes controlled by undernutrition, Allen[4] has regularly found marked hyperglycemic curves following protein ingestion. Mosenthal, Clausen and Hiller[5] found that carbohydrate-free meals cause no hyperglycemia in non-diabetics or in some diabetics, but in other diabetics they produce more or less rise of blood sugar. This rise was found particularly in cases with initially low blood sugars, and was commonly missed in cases with marked hyperglycemia at the outset. The rise of blood urea after protein ingestion in rela-

---

[1] Jacobsen, A. Th. B.: Biochem. Ztschr., 1913, lvi, 471–494.

[2] Biochem. Ztschr., xlix, 1913, 278–292.

[3] Bull. Johns Hopkins Hospital, xxvi, 1915, 211–215.

[4] Am. Jour. Med. Sc., 1917, p. 153, Chart VIII.

[5] Arch. Int. Med., xxi, 1918, 93–108.

TABLE I.—50 GM. PROTEIN TEST IN FIVE NORMAL PERSONS.

| | 1 Mr. H. E., 22 years. | | | | 2 Dr. S., 29 years. | | | | 3 Dr. J., 34 years. | | | 4 Miss M., 28 years. | 5 Mr. C., 22 years. | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Blood. | | Urine. | | Blood. | | Urine. | | Blood urea, mgm. per 100 c.c. | Urine. | | Blood urea, mgm. per 100 c.c. | Blood urea, mgm. per 100 c.c. | Urine. | |
| | Plasma sugar, per cent. | Urea, mgm. per 100 c.c. | Urea, per cent. | Urea, gm. | Plasma sugar, per cent. | Urea, mgm. per 100 c.c. | Urea, per cent. | Urea, gm. | | Urea, per cent. | Urea, gm. | | | Urea, per cent. | Urea, gm. |
| Before . . . . | 0.106 | 20.0 | 2.63 | .. | 0.110 | 13.5 | 1.71 | .. | 24.2 | 2.42 | .. | 15.0 | 46.4 | 2.39 | |
| 2 hours . . . | 0.100 | 21.9 | 1.81 | 1.46 | 0.125 | 18.3 | 1.30 | 1.69 | .. | 2.13 | 2.13 | 20.5 | 42.1 | | |
| 4 hours . . . | 0.114 | 28.0 | 2.16 | 1.49 | 0.115 | 19.4 | 1.39 | 2.26 | 28.0 | 2.29 | 2.68 | .. | 44.2 | 2.74 | 6.44 |
| 6 hours . . . | 0.105 | 28.0 | 2.38 | 1.45 | 0.115 | 22.6 | 0.64 | 2.18 | 32.6 | 1.75 | 1.96 | 31.4 | 46.4 | | |
| 8 hours . . . | .. | 26.0 | .. | .. | .. | 17.2 | 0.56 | 1.81 | 28.4 | 1.82 | 1.86 | .. | 46.4 | 3.83 | 6.05 |
| 24 hours . . . | .. | 18.0 | 1.02 | 10.62 | .. | 20.5 | 1.14 | 6.65 | 22.2 | 1.25 | 10.00 | 17.2 | 38.8 | 2.80 | 13.55 |
| Urea excreted in 24 hours (gms.) | .. | .. | .. | 15.04 | .. | .. | .. | 14.16 | .. | .. | 18.65 | .. | .. | .. | 26.05 |

tion either to retention or metabolic variations has been investigated, especially by Addis and Watanabe.[6] An investigation was therefore undertaken to determine the curve of plasma sugar in normal, diabetic and nephritic individuals after standard protein meals and was extended to cover the urea of blood and urine.

METHODS. The standard procedure was that the subjects voided urine and were bled, then ate a breakfast consisting solely of 50 gm. protein in the form of beefsteak, and 200 c.c. water. Two patients in the series received only 40 gm. protein. Every two hours thereafter urine was passed and blood taken and 200 c.c. water drunk. After eight hours a very light supper was taken of cornstarch and candy with a trifle of fruit, having negligible protein content. Water was then taken at will, but no other food until after the blood and urine samples the next morning, which closed the twenty-four-hour period. Sugar was determined according to Benedict, urea by the urease method of Van Slyke and Cullen and total nitrogen by the usual Kjeldahl method. Blood sugars were determined in two of the normal individuals, in confirmation of the writer's previous work regarding the negative effect of protein. The sugar changes were within the limits of accidental fluctuation.

There was a very appreciable rise of the blood urea during the eight-hour experimental period, followed by a fall at the end of the twenty-four hours in every individual except No. 5. This person was a technician in the laboratory. His urine, like that of all the others in this group, was free from albumin and casts. He was supposedly in perfect health, though subject to occasional headaches. It was learned that he had by chance eaten excessive quantities of meat on the day before the test. These facts explain the exceptionally high excretion and the lower blood urea after twenty-four hours. In a strict sense this person probably has a slight nephritis, and he has been advised to follow a fairly low protein diet. Irrespective of this question it is evident that the initial percentage of blood urea is not a safe criterion of the tendency to rise after a certain protein intake.

TABLE II.—PROTEIN TESTS IN SEVEN DIABETICS.

1.—28 years.

(40 gm. protein.)

| | Blood. | | |
|---|---|---|---|
| | Urea, mgm. per 100 c.c. | Plasma sugar, per cent. | Chlorides, mgm. per 100 c.c. |
| Before | 20.0 | 0.139 | 568 |
| 1 hour | 24.0 | 0.174 | 518 |
| 2 hours | 26.0 | 0.174 | 576 |
| 4 hours | 32.2 | 0.158 | 576 |
| 6 hours | 36.2 | 0.165 | 587 |
| 9 hours | 35.2 | 0.123 | 572 |
| 24 hours | 29.0 | 0.122 | |

Excreted 11.6 grams total nitrogen.

---

[6] Arch. Int. Med., 1917, xix, 507–517.

2.—27 years.

(40 gm. protein.)

| | Blood. | | | Urine. | | |
|---|---|---|---|---|---|---|
| | Urea, mgm. per 100 c.c. | Plasma sugar, per cent. | Chlorides, mgm. per 100 c.c. | Vol., c.c. | Sugar. | Acetone. |
| Before | 47.2 | 0.043 | 520 | .. | 0 | 0 |
| 1 hour | 53.4 | 0.070 | 557 | | | |
| 2 hours | 60.4 | 0.068 | 504 | 85 | 0 | 0 |
| 4 hours | 74.6 | 0.064 | 527 | 290 | 0 | 0 |
| 6 hours | 76.6 | 0.050 | 546 | 240 | 0 | 0 |
| 9 hours | 58.4 | 0.043 | 521 | 290 | 0 | 0 |
| 24 hours | 54.4 | 0.040 | .. | 1700 | 0 | 0 |

Excreted 14.2 grams total nitrogen.

3.—35 years.

| | Blood. | | | Urine. | | |
|---|---|---|---|---|---|---|
| | Urea, mgm. per 100 c.c. | Plasma sugar, per cent. | Chlorides, mgm. per 100 c.c. | Vol., c.c. | Sugar. | Acetone. |
| Before | .. | 0.074 | 587 | | | |
| 1 hour | 40.0 | 0.110 | 584 | 260 | 0 | 0 |
| 3 hours | 42.0 | 0.144 | 590 | 145 | 0 | Slight |
| 5 hours | .. | 0.139 | 555 | 115 | 0 | Mod. |
| 7 hours | 44.0 | 0.136 | 590 | 125 | 0 | Faint |
| 9 hours | 56.0 | 0.091 | 587 | 130 | 0 | 0 |
| 24 hours | 50.4 | 0.048 | 561 | | | |

4.—64 years.

| | Blood. | |
|---|---|---|
| | Plasma sugar, per cent. | Urea, mgm. per 100 c.c. |
| Before | 0.176 | 24.0 |
| 2 hours | 0.197 | 26.0 |
| 4 hours | 0.205 | 30.0 |
| 6 hours | 0.205 | 34.2 |
| 24 hours | 0.242 | 39.2 |

5.—43 years.

| | Plasma sugar, per cent. |
|---|---|
| Before | 0.130 |
| 5 minutes after | 0.130 |
| 1 hour after | 0.144 |
| 2 hours | 0.181 |
| 3½ hours | 0.192 |
| 4½ hours | 0.208 |
| 5½ hours | 0.203 |
| 6 hours | 0.203 |

6.—31 years.

| | Blood. | | | Urine. | | | | |
|---|---|---|---|---|---|---|---|---|
| | Urea mgm. per 100 c.c. | Plasma sugar per cent. | $CO_2$ vol. per cent. | Vol. c.c. | Urea per cent. | Urea gm. | Sugar. | Acetone. |
| Before | 31.4 | 0.091 | 61.4 | .. | 1.79 | .. | 0 | 0 |
| 2 hours | 35.8 | 0.135 | 68.1 | 87 | 2.23 | 1.96 | 0 | 0 |
| 4 hours | 46.4 | 0.147 | 62.5 | 113 | 2.24 | 2.55 | 0 | 0 |
| 6 hours | 46.4 | 0.132 | 57.8 | 103 | 2.80 | 2.89 | 0 | 0 |
| 8 hours | 46.4 | 0.125 | 70.0 | 100 | — | — | — | — |
| 24 hours | 34.6 | 0.150 | — | 1020 | 1.49 | 15.21 | 0 | 0 |

Excreted 22.6 grams urea and 14.5 grams total nitrogen.

7.—27 years.

| | October 26. | | | | | | | | | November 20. | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Blood. | | | | Urine. | | | | | Blood. | | Urine. | | | | |
| | Urea, mgm. per 100 c.c. | Plasma sugar, per cent. | Chlorides, mgm. per 100 c.c. | $CO_2$ vol., per cent. | Vol., c.c. | Urea, per cent. | Urea, gm. | Sugar, per cent. | Nitroprusside. | Urea mgm. per 100 c.c. | Plasma sugar, per cent. | Vol., c.c. | Urea, per cent. | Urea, gm. | Sugar, per cent. | Nitroprusside. |
| Before . | 14.8 | 0.314 | 582 | 51.0 | .. | 1.38 | .. | 4.8 | Heavy | 11.8 | 0.194 | — | 0.46 | .. | 0 | 0 |
| 2 hours . | 17.8 | 0.341 | 560 | 45.3 | 185 | 1.23 | 2.27 | 5.5 | " | 14.0 | 0.191 | | | | | |
| 4 hours . | 18.8 | 0.341 | 558 | 40.4 | 200 | 1.01 | 2.02 | 5.5 | " | 20.5 | 0.272 | 258 | 1.02 | 2.64 | 0 | 0 |
| 6 hours . | 16.6 | 0.319 | 566 | 43.3 | 248 | 1.36 | 3.36 | 3.66 | " | 24.7 | | | | | | |
| 8 hours . | 17.8 | 0.280 | 574 | .. | 225 | 1.23 | 2.76 | 2.0 | " | 21.6 | .. | 190 | 1.78 | 3.39 | 0 | 0 |
| 24 hours . | 9.0 | — | — | 47.1 | 1780 | 0.59 | 10.50 | 0.65 | " | 20.5 | 0.269 | — | 0.87 | .. | 0 | 0 |

Excreted 20.9 gm. urea.

(Total excretion lost.)

The diabetics were mostly young, but were all alike in having the severe form of the disorder, more or less completely under control by undernutrition treatment. There was a history of three to ten years' duration and an advanced degree of emaciation and weakness in all except No. 1, who had apparently been diabetic less than six months and was only slightly thin. The preceding diets ranged from fasting, in the case of the first test on patient No. 7, up to 60 gm. protein, 20 gm. carbohydrate and 1000 calories in patient No. 3. All were free from albuminuria or casts except No. 2. There was moderate edema in No. 2 and slight in No. 6 at the time of the test. Patient No. 3 had formerly been subject to edema, but was free at this time. The others had never exhibited edema. The plasma chlorides remained within normal limits in all determinations. The blood-pressure was normal or subnormal in all.

The general program of the tests was the same as above described, except that after the initial period of eight or nine hours a supper of negligible food value was given, consisting of bran biscuits, agar jelly, one dish of thrice-cooked vegetable, and coffee or infusion of cocoa shells. Certain points concerning the results in individual patients may be noted as follows:

Patient No. 1, starting with a normal blood urea of 20 mgm. per 100 c.c., showed a greater rise than any of the normal subjects, viz., to 36.2 mgm. at the sixth hour. The plasma sugar, starting with a slight hyperglycemia of 0.139 per cent., jumped to its maximum of 0.174 per cent. in one hour, and in general fell after the second hour. Undernutrition on this day is indicated by the plasma sugar, lower after twenty-four hours than at the outset, and by the excretion of nitrogen exceeding the intake. This absence of retention is of interest in connection with the prolonged rise of blood urea.

Patient No. 2 was admitted with a history not only of the severest type of diabetes but also of several mild attacks of renal colic followed by passage of small calculi, with blood in the urine at these times and pus afterward. In the hospital the albumin in his urine frequently gave Esbach readings as high as 1.5; the sediment showed enormous numbers of pus cells but no casts. Later attacks caused his blood urea to rise above 130 mgm. per 100 c.c., and at the time of the test he may be considered as representing a combination of diabetes and nephritis. Starting at 47.2 mgm. per 100 c.c., the blood urea rose to its maximum of 76.6 mgm. at the sixth hour. At the same time the twenty-four-hour nitrogen output was nearly double the intake, in correspondence with the very low diet in a patient possessing a minimum of body fat. In the days preceding the test, the rather rapid fall of the formerly intractable blood sugar to a subnormal level was another indication of danger. Under these circumstances, though the diabetes was the most desperately severe of the series, the rise of the plasma sugar from the ingestion of protein was considerable in proportion to the original amount but trivial in absolute figures. The patient is still alive after nine months, free from glycosuria and acidosis and much improved in strength, and at home with normal plasma sugar.

Patient No. 3, with a somewhat less degree of diabetes and undernutrition, started with plasma sugar of 0.074 per cent., and reached the maximum of 0.144 per cent. in three hours after eating. Thereafter the curve progressively fell. The urea analysis before eating was lost. From 40 mgm. per 100 c.c. one hour after eating, it rose to 56 mgm. at the ninth hour. The lack of parallelism with the sugar curve is especially noticeable. During digestion a well-marked acetone reaction developed in the urine and later disappeared, due either to the protein or to the trifle of fat in the lean meat.

Patient No. 4 was tested when the diabetes was only partially under control, as indicated by the hyperglycemia of 0.176 per cent. This rose to its maximum of 0.205 per cent. at the fourth and sixth hours after eating and traces of sugar were excreted. The urea curve was parallel, starting at 24 mgm. per 100 c.c. and reaching 34.2 mgm. at the sixth hour. The morning figures of 0.242 per cent. plasma sugar and 39.2 mgm. urea proved that the patient broke diet in the evening.

Patient No. 5 had an unusually intractable diabetes for his age, and the question arose whether his slow progress was due to breaking diet or whether his hyperglycemia could actually be due to the small quantities of prescribed food. He was accordingly given the 50 gm. protein test while under constant observation in the laboratory, and the blood followed for sugar alone for six hours. The marked hyperglycemia demonstrated the true severity of the condition, and subsequent acquaintance with the patient and the ultimate favorable outcome confirmed his fidelity.

Patient No. 6 underwent fasting immediately upon admission and was given the test with 50 gm. protein as soon as the plasma sugar became normal. The plasma sugar rose to a maximum of 0.147 per cent. at the fourth hour, but was still 0.15 per cent. the next morning, indicating that the patient could not yet tolerate this quantity of protein without hyperglycemia. For this reason the protein ration for the ensuing days was set at 30 gm. During digestion the $CO_2$ capacity of the plasma showed some of the familiar digestive variations. The blood urea rose from 31.4 mgm. per 100 c.c. to 46.4 mgm. at the fourth hour and held this maximum for at least four hours. The two-hour urinalyses showed no retention as compared with normal persons in Table I and the total twenty-four-hour excretion was excessive, as is natural for the emaciated organism under these conditions. No acetonuria appeared during the test.

Patient No. 7 was subjected to two tests, the first during active glycosuria before treatment, the second during the early stage of treatment, when there was hyperglycemia but no glycosuria.

In the first test on October 26 the variations in blood urea and sugar were slight and parallel for the first eight hours. This test represented a partial fast following abruptly upon the former mixed diet; therefore the result at the end of eight hours was a slight fall in blood sugar. The blood urea, on the contrary, was a trifle higher than at the first, but after twenty-four hours had fallen to the markedly low level of 9 mgm. The urea excretion was high, both for the twenty-four-hour and for the two-hour periods. The plasma bicarbonate fell distinctly during this protein test and the urinary nitroprusside reaction remained heavy. At the same time the urinary sugar fell.

By November 20 the patient had reached a point where sugar and nitroprusside reactions were constantly negative in the urine and the plasma bicarbonate normal, but hyperglycemia persisted. The blood urea started at 11.8 mgm. per 100 c.c., reached a maximum of 24.7 mgm. at the sixth hour and was still up to 20.5 mg. the next morning. The plasma sugar was 0.194 per cent. before eating, practically the same two hours after, but at the fourth hour was found to be 0.272 per cent. and was still 0.269 per cent. the next morning. The reason for the elevation of both urea and sugar here after 50 gm. protein probably is that the preceding diet had contained only 30 gm. protein. The quantitative twenty-four-hour urine collection was spoiled by the patient's fault. Though this patient's phenolsulphonephthalein test had shown an elimination of 60 per cent. the first hour and 20 per cent. the second hour, and both the urea and chloride functions had likewise been found normal, the impermeability for sugar is indicated by the absence of glycosuria with the high plasma sugars mentioned.

The first three patients listed had typical hypertensive nephritis of severe grade, with only traces of albumin and rare casts. No. 1 had slight edema and ascites, due to myocarditis and cirrhosis of the liver. Nos. 2 and 3 were free from edema. Case No. 4 was of the "parenchymatous" type, with normal or subnormal blood-pressures, extremely heavy albumin and much edema. All had been for some time preceding on diets liberal in calories but containing only 30 or 40 gm. protein. The program of the test days was as described for normal subjects. The liberal supply of water of these days was a sharp departure from the usual regimen of these patients, who had been on diets poor in both salt and fluid, and instead of sweeping out nitrogen it may have contributed to its retention.

All the tests were characterized by marked deficiency of the urea output as compared with normals, and especially in Case 1 this apparent retention was extreme. In all except Case 4 the urine volume indicated also a marked retention of fluid. The blood urea rose in all during the first eight hours, but this rise was nothing

TABLE III.—50 GM. PROTEIN TEST IN FOUR NEPHRITICS.

| | 1.—42 years. | | | | | | | | 2.—40 years. | | | | | | 3.—45 years. | | | | 4.—19 years. | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Blood. | | | Urine. | | | | | Blood. | | | Urine. | | | Blood | Urine. | | | Blood. | Urine. | | |
| | Urea mgm. per 100 c.c. | Plasma sugar per cent. | Chlorides mgm. per 100 c.c. | Vol. c.c. | Urea per cent. | Urea gm. | Chlorides per cent. | Chlorides gm. | Urea mgm. per 100 c.c. | Plasma sugar per cent. | Chlorides mgm. per 100 c.c. | Vol. c.c. | Urea per cent. | Urea gm. | Urea mgm. per 100 c.c. | Vol. c.c. | Urea per cent. | Urea gm. | Urea mgm. per 100 c.c. | Vol. c.c. | Urea per cent. | Urea gm. |
| Before | 16.0 | 0.108 | 508 | .. | 0.49 | .. | 0.19 | .. | 24.6 | 0.146 | 585 | .. | .. | .. | 31.5 | .. | 0.71 | .. | 10.8 | | | |
| 2 hrs. | 19.0 | 0.133 | 498 | 21.5 | 0.24 | 0.05 | 0.05 | 0.01 | 26.0 | 0.158 | 585 | .. | .. | .. | 40.6 | 34 | 1.07 | 0.36 | 12.9 | 35 | 0.83 | 2.92 |
| 4 hrs. | 28.0 | 0.143 | 498 | .. | .. | .. | .. | .. | 33.6 | 0.144 | 543 | 51 | 1.82 | 0.93 | 32.3 | 73 | 1.04 | 0.76 | 10.2 | 105 | 1.79 | 1.88 |
| 6 hrs. | 34.6 | 0.132 | 480 | 57.0 | 0.54 | 0.31 | 0.04 | 0.02 | 40.0 | 0.136 | 620 | .. | .. | .. | 35.7 | 62 | 1.03 | 0.64 | 16.2 | 125 | 1.49 | 1.86 |
| 8 hrs. | 36.2 | 0.106 | 444 | 36.0 | 0.69 | 0.24 | 0.10 | 0.02 | 37.8 | 0.139 | 593 | 173 | 1.99 | 2.25 | 34.3 | 25 | 1.35 | 0.33 | 20.5 | 65 | 1.69 | 1.10 |
| 12 hrs. | 30.1 | .. | 462 | | | | | | | | | | | | | | | | | | | |
| 24 hrs. | 27.0 | .. | 523 | 750 | 0.25 | 1.90 | 0.27 | 2.02 | .. | .. | .. | 275 | 1.47 | 4.04 | 34.3 | 660 | 0.61 | 4.04 | 10.8 | 1410 | 0.47 | 0.64 |
| Urea excreted in 24 hrs. gm. | | | | | | 2.46 | | | | | | | | 7.23 | | | | 6.15 | | | | 11.79 |

extraordinary as compared with the records of the normals and diabetics and failed totally to correspond to the comparative nitrogen elimination. In Case 1 the blood urea remained elevated at the end of twenty-four hours, as if indicating the nitrogen retention, but in Case 4 it ended at the same level where it began, and in Case 3 the elevation was trivial. Dilution probably played some part in maintaining the percentages at such levels; also, with the ample carbohydrate and calories of the usual diets, true storage as protein may have been a factor; but obviously much is unknown concerning the nitrogen retention of nephritis.

Patient No. 1 started with a normal plasma sugar of 0.108 per cent., which was moderately increased during the first six hours after eating. Patient No. 2 started with hyperglycemia of 0.146 per cent., which rose to 0.158 per cent. at the second hour, then gradually fell. Several previous writers have called attention to hyperglycemia in occasional cases of nephritis, especially with hypertension, and as such reports have been based on various methods of analysis, it is evident that the excess material present affects copper salts as well as picric acid, and may be glucose. But in view of the possible errors due to creatinin or similar substances it was deemed best to defer any conclusions concerning nephritic blood sugars until truer figures should be available by the use of the procedure recently proposed by Benedict[7] for removal of the interfering material.[8] One of us (J.) proposes to carry out further tests comparing the Benedict method with a copper reduction test for the blood sugar.

Conclusions. 1. After a standard meal of 50 gm. protein there is generally a rising curve of urea in the blood, which, as a rule, is higher in patients with severe diabetes or nephritis than in normal persons.

2. The blood-urea level is not a reliable index of nitrogen retention. In some instances, for example in diabetics, it may possibly bear some relation to the rate of protein catabolism. On the whole, it seems to be governed by so many factors of the previous diet, fluid retention and unknown metabolic conditions that by itself it forms an uncertain basis for conclusions.

3. The plasma sugar of normal persons is not appreciably altered by this quantity of protein.

4. The substances in nephritic blood, which react like sugar to the Benedict test, are increased after a protein meal.

5. A typical curve of hyperglycemia follows the ingestion of protein in suitably severe cases of diabetes. This is likely to be missed

---

[7] Benedict, S. R.: Paper read before the Biochemical Society, Cincinnati, Ohio, December 30, 1919.

[8] Subsequent analyses in this clinic with charcoal kindly provided by Dr. Benedict have shown no more than slight changes in the above curves. Therefore either the rise of plasma sugar after protein meals is not specific to diabetes or a diabetic tendency is thus demonstrable in some cases of nephritis.

if active symptoms of glycosuria or hyperglycemia are present, as the effect of 50 gm. protein could scarcely be expected to be noticeable amid such a surplus of sugar. Rarely also the rise may be slight in extreme exhaustion. Under the conditions of strict diet treatment this reaction to a standard protein meal may be used as a measure of the severity of diabetes and a guide both to the protein allowance and the total ration, and it has lately been used for these purposes in this clinic.

---

# TREATMENT OF SURGICAL SHOCK IN THE ZONE OF THE ADVANCE.[1]

By W. Richard Ohler, M.D.,
FORMERLY MAJOR, M.C., BASE HOSPITAL NO. 7, BOSTON CITY HOSPITAL, BOSTON, MASS.

The nature and treatment of shock has, in recent years, attracted considerable attention from the profession, especially on the part of the surgeon. War-time conditions naturally brought under observation greatly increased numbers of shock cases. The situation presented exceptional opportunities for the study of shock and it also demanded a plan for specialized treatment. This was all the more necessary when it became evident that in the advance zones the surgeons, when busy, were very busy, and had no time for carrying out specialized treatment. Internists, assisted and trained by physiologists, were therefore called in to help in what has generally been considered a strictly surgical problem. The plan devised by the medical department of the A. E. F. was to send into the advanced areas so-called "shock" teams, composed of medical officers (internists), nurses and corpsmen, especially trained and equipped for the treatment of shock. In preparation for the work the medical officers were given a special course in the nature and treatment of shock at the central medical laboratory at Dijon, under the supervision of Lieut.-Col. Walter B. Cannon, M.C., Boston, Mass.

In general the substance of the lectures and demonstrations given at Dijon may be summarized as follows:

I. Theoretical. Whether shock be due to hemorrhage, exposure to cold and wet, lack of food and drink, injury to muscle or nerve tissues, singly or in combination, it was agreed that in shock we find a condition of persistent low arterial blood-pressure, rapid pulse, pallor or slight cyanosis, sweating and rapid respiration, slight or

---

[1] The cases reported were observed by Shock Team No. 116, in charge of Captain George Strode, M.C. (Harrisburg, Pa.), and Shock Team No. 154, in charge of Captain W. R. Ohler, M.C., during the St. Mihiel and Argonne Meuse offensives. I wish to express my appreciation to Dr. Strode for permitting me to group all the cases together.